

## Review on the Incidence and Pathology of Infectious Bursal Disease

*Tsegaye Kegne and Mersha Chanie*

University of Gondar, Faculty of Veterinary Medicine,  
Department of Veterinary Paraclinical Studies, P.O. Box, 196, Gondar, Ethiopia

**Abstract:** Infectious bursal disease (IBD) is caused by a virus that is a member of the genus *AviBirnavirus* of the family *Birnaviridae*. Although turkeys, ducks, guinea fowl and ostriches may be infected, clinical disease occurs solely in chickens. Only young birds are clinically affected. Severe acute disease of 3-6-week-old birds is associated with high mortality, but a less acute or subclinical disease is common in 0-3-week-old birds. This can cause secondary problems due to the effect of the virus on the bursa of fabricius. IBD virus (IBDV) causes lymphoid depletion of the bursa and if this occurs in the first 2 weeks of life, significant depression of the humoral antibody response may result. Two serotypes of IBDV are recognized; these are designated serotypes 1 and 2. Clinical disease has been associated with only serotype 1 and all commercial vaccines are prepared against this serotype. Very virulent strains of classical serotype 1 are now common and are causing serious disease in many countries. Clinical disease due to infection with the IBDV, also known as Gumboro disease, can usually diagnosed by a combination of characteristic clinical signs and post-mortem lesions. Laboratory confirmation of disease, or detection of subclinical infection, can be carried out by demonstration of a humoral immune response in unvaccinated chickens or by detecting the presence of viral antigen or viral genome in tissues. In the absence of such tests, histological examination of bursae may be helpful. Death of chickens usually starts at the 30th day of age and continues to the 5th day after infection and with falling spiking curve. In Ethiopia IBDV is frequently reported from Debre-Zeit andassa, Kombolcha and Gondar.

**Key words:** Infectious Bursal Disease • Gross Lesion • Histopathology • Host • Occurrence

### INTRODUCTION

Infectious Bursal Disease (IBD) is caused by an acute, highly contagious *Birnavirus* that results in mortality and immunosuppressant in young chickens. Since its original isolation in Gumboro, Delaware, the disease has inflicted profound economic losses on the poultry industry worldwide. Infectious bursal disease virus (IBDV) and other *Birnaviruses* are single-shelled, non-enveloped viruses that contain a bi-segmented, double-stranded RNA genome [1].

The infectious bursal disease (IBD) is recognized as an important disease of young chickens worldwide. It causes unthriftiness, anorexia, ruffled feathers, diarrhoea and mortality in affected flocks. The infectious bursal disease virus (IBDV) infection of chickens less than three weeks of age causes immunosuppression with increased a susceptibility to other diseases and lack of humoral response to vaccinations [2].

Although chickens are highly susceptible to IBD, other poultry species such as turkeys and ducks show minimal or no susceptibility to the clinical disease under natural conditions. Serological evidence of infection has been established in turkeys, even though neither of the two IBDV serotypes has produced clinical disease in this species. In addition, IBDV has been isolated from clinically healthy ducks which were negative for IBD antibody. Thus, these three poultry species appear to have different levels of susceptibility to IBDV infection, being highest in chickens [3].

Chicken production under back yard system has long been practiced in Ethiopia. Constraints which could restrict the potentiality of village in Ethiopia include low input of feed, poor management, infectious disease and lack of appropriate selection and breeding practice. Recently, the occurrences of IBD in some parts of the country have wiped out large number of chicken in private commercial and government owned poultry farms [4].

**Corresponding Author:** Mersha Chanie, University of Gondar, Faculty of Veterinary Medicine,  
Department of Veterinary Paraclinical Studies, P.O. Box, 196, Gondar, Ethiopia.

The objectives of this seminar paper are therefore to summarize the occurrence and incidence of IBDV and to discuss gross and microscopic lesions of IBD.

**Infectious Bursal Disease Virus:** Infectious bursal disease (IBD) was first recognized as a distinct clinical entity in 1957. Initially the malady described as “avian nephrosis” on account of tubular degenerative lesions found in the kidneys of infected broiler chickens [5].

The disease is also known as “Gumboro disease” since the first outbreaks occurred in and around the area of Gumboro, Delaware, USA. Predominant signs of illness included trembling, ruffled feathers, watery diarrhoea, anorexia, depression, severe prostration and death. In addition, hemorrhages in the thigh and leg muscles, increased mucus in the intestine, liver infarction, renal damage and enlargement of the bursa of Fabricius were lesions commonly observed at necropsy. Early studies suggested that the causative agent was a nephron-pathogenic strain of infectious bronchitis virus due to similar gross changes observed in the kidney [2].

Subsequent studies however, revealed that IBV immunized birds could still be infected with the “infectious bursal agent” (IBA) and developed changes in their cloacal bursas specific for the disease. Following successful isolation of IBA in embryonated chicken eggs [2] proposed that the disease be termed “infectious bursal disease” due to its pathognomonic bursal lesions. The immunosuppressive effects of infectious bursal disease virus (IBDV) infections were first disclosed. These factors, along with the high tendency for IBD infections to recur in successive flocks, emphasized the need for stringent measures of prevention and control. Prior to 1984, spread of both the clinical and subclinical forms of the disease was satisfactorily controlled by vaccination programs. However, in 1984 and 1985, a significant increase in mortality, condemnations and vaccine failures were reported in the Delmarva Peninsula broiler growing area. These newly emergent viruses were capable of breaking through maternal immunity against classic strains of IBDV. *In vivo* reciprocal cross-challenge tests showed that unlike classic or standard strains of IBDV, the field isolates caused rapid atrophy and minimal inflammation of the cloacal bursa when inoculated into susceptible leghorns. Studies suggested that a major antigenic shift in serotype I viruses had occurred in the field [6].

The IBDV field isolates were characterized as antigenic “variants” of serotype 1 IBDV, while the older serotype 1 viruses discovered prior to these newly

emergent viruses were called classical strains of IBDV. Currently in the United States, clinical cases are rarely reported and these variant strains are the predominant viruses circulating in the field. Outbreaks of very virulent IBDV (vvIBDV) were first reported in Europe in 1987-1988. Highly virulent IBDV (vvIBDV) infections are characterized by a peracute onset of severe clinical disease and high mortality. Although these new serotype 1 viruses demonstrate increased virulence in their ability to break through the existing level of maternal immunity; they are antigenically similar to the classic strains of IBDV. Strains of vvIBDV have rapidly disseminated to every poultry-producing country, except Canada, Mexico, Australia and New Zealand [7].

**Etiology of IBD:** Infectious bursal disease virus (IBDV) is classified as a member of the *Birnaviridae* family. The family includes 3 genera: *Aquabirnavirus* whose type species is infectious pancreatic necrosis virus (IPNV), which infects fish, mollusks and crustaceans; *Avibirnavirus* whose type species is infectious bursal disease virus (IBDV), which infects birds; and *Entomobirnavirus* whose type species is *Drosophila* X virus (DXV), which infects insects. Viruses in this family possess bi-segmented, double-stranded RNA (dsRNA) genomes, which are packaged into single-shelled, non-enveloped virions. The capsid shell exhibits icosahedral symmetry composed of 32 capsomeres and a diameter ranging from 55 to 65 nm [7].

**Variant Strains of IBD:** Two distinct serotypes of infectious bursal disease virus (IBDV) are known to show clinical disease in chickens younger than 10 weeks. Older chickens usually show no clinical signs. Antibodies are sometimes found in other avian species, but no signs of infection are seen. Serotype 2 antibodies are very widespread in turkeys and are sometimes found in chickens and ducks. There are no reports of clinical disease caused by infection with Serotype 2 virus [8].

Control of IBD has been further complicated by the recognition of variant strains of the IBD virus. Variant viruses induce damage in the BF in chickens, even when high and uniform antibody titers are present. Variant strains do not cause obvious clinical disease, but immune-suppression. In Chicken affected by classical IBDV the bursa of Fabricius undergo rapid atrophy (lymphocyte depletion) without inflammatory changes observed early in the infection. These variants are not from a different serotype, but are antigenically different enough to cause immunosuppression problems [9].

Often IBD is a serious problem in integration and losses occur despite persistent efforts at reducing field virus's exposure through a biosecurity program, maintenance of adequate and uniform maternal titers and an effective broiler vaccination program. Consideration should be given to vaccinating breeders with inactivated vaccines containing standard and variant strains of the IBD virus occurs [10].

**Epidemiology:** Incidence and distribution: Birds that are 3-6 weeks age are the most susceptible to clinical disease. The IBDV is susceptible to mutation, highly resistant to heat and chemicals and can persist in faces, bedding, contaminated feed and water for up to four months in certain conditions. Mode of transmission is primarily through fecal oral route, with aerosol spread considered to be less important. There is no evidence that IBD can be transmitted in embryos or semen [11].

The viral incubation period is about 2-3 days and can be shed as soon as 24 hours following infection and can last up to two weeks. The disease is highly contagious, can spread through the movement of poultry products, equipment, feed bags, vehicles and people and to a lesser extent, through aerosols of dust. Transmission of IBD between wild birds and poultry is likely to be due to scavenging of dead chickens, ingestion of contaminated water, or exposure of respiratory or conjunctiva membranes to contaminated poultry dust [11, 12].

Classical serotype 1 IBD infection in wild birds is believed to be subclinical. Recent research shows that wild birds play a role in the epidemiology of IBDV by acting as a reservoir for the virus. Classical serotype 1 IBDV strains are endemic throughout the world. Very virulent IBD is endemic in parts of southern Asia, Indonesian island region, South America, Middle East and Africa [9].

Serotype 1 IBDV antibodies have been detected in Australian wild birds including carrion crows and rock pigeons which were found around barns and domestic chicken flocks. There is no data that suggest IBDV is transmitted by wild birds in Australia, however direct or indirect transmission of the virus between wild birds and domestic chickens probably may occur. It is strongly believed that the serotype IBDV 1 is highly host specific to chickens. However, IBDV has been isolated from a sparrow in China, which suggests that wild birds may have an important role in the natural history of IBDV. Reports have shown that serotype 2 of IBDV is more prevalent in many species of wild birds, with the natural host considered to be turkeys [12].

**Hosts:** The natural hosts of IBDV are the domestic fowl including chickens and turkeys. Other wild bird like healthy ducks, guinea fowl, quail and pheasants have been found to be naturally infected by serotype 1 IBDV. There is no evidence that IBD virus can infect other animals, including humans [13].

**Transmission of IBD Virus:** Chickens infected with the IBD virus shed the virus in their feces. Feed, water and poultry house litter become contaminated. Other chickens in the house become infected by ingesting the virus [8]. The lesser mealworm (*Alphitobus diaperinus*) has been shown to carry the virus. Because of the resistant nature of the IBD virus, it is easily transmitted mechanically among the farms by people, equipment and vehicles [14].

**Disease Status in Ethiopia:** In Hailu's *et al.* [15] study, the incidence of IBD was found 38.39% in Bahir Dar and 17.40 % in Farta. The case fatality rates were 98.56% in Bahir Dar and 77.73% in Farta. There was significant difference ( $p < 0.05$ ) in the incidence and case fatality rates of IBD among the study districts. Chickens in the households of Bahir Dar district was found 1.69 (OR=1.69) times more likely to be affected by IBD than those in Farta district. Birds with a clinical signs of vent picking and diarrhea had gross and microscopic lesions suggestive of IBD. Agar gel Immuno-diffusion test revealed the presence of antibodies against IBD in the serum of most recovered birds from IBD. Again [15] report indicated that IBD in village chickens in Ethiopia. Thus, it is of paramount importance to design cost effective control methods against IBD in order to improve the productivity and welfare of village chickens and also to conserve the indigenous chicken genetic resource.

Outbreak of suspected IBD case was also reported to the National Veterinary Institute from a poultry farm in Debre-Zeit town located 45 kms to the south of Addis Ababa as indicated by Zeleke *et al.* [17]. Two nearby poultry farms were also affected by the outbreak. The farms share a common feed mill and vehicles. At the onset of the outbreak there were about 40,000 broilers and 10,000 layers.

Both farms import day old chicks from the Netherlands and Egypt. Other farms in the same area import day old chicks from Kenya, South Africa and Germany [18]. Case report study from Andassa poultry farm indicate that the overall mortality of chicken due to IBD was 12% in young (1-70 day old) and 7% in adult (>70 day old) and 100% seroprevalence has been recorded in non-vaccinated flock [19].

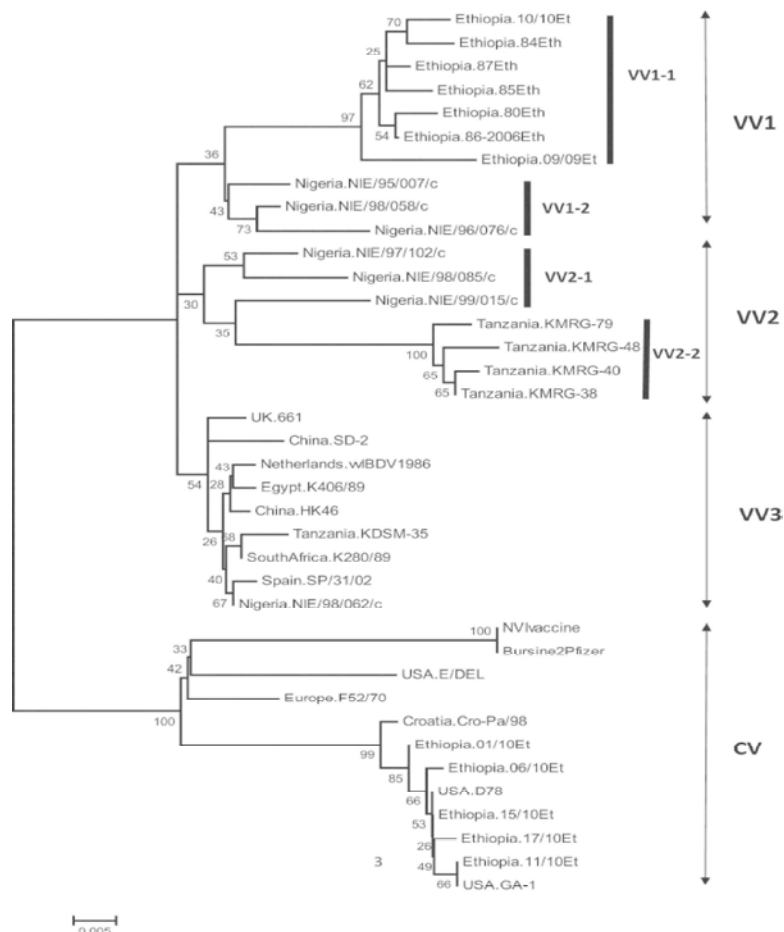


Fig 1: Genetic characterization of the Ethiopian Isolates of IBDV. Source: Jenberie *et al.* [16]

Jenberie *et al.* [16] reported that phylogenetically, Ethiopian IBDVs represented two genetic lineages: very virulent (vv) IBDVs or variants of the classical attenuated vaccine strain (D78). The nucleotide identity between Ethiopian vvIBDV s ranged between 0% and 2.6%. Ethiopian vvIBDV s are clustered phylogenetically with the African IBDV genetic lineage, independent of the Asian/European lineage. This report demonstrates the circulation of vvIBDV in commercial and breeding poultry farms in Ethiopia. Kassa and Molla [20] reported that the seroprevalence of IBD in districts found in West Gojam zone of Amhara region, Ethiopia ranges from 72-78%. The lowest and the highest seroprevalence were recorded from Meshenti and Andassa study sites of this zone respectively. The proportion of seropositive chickens, however, doesn't vary significantly among the three districts.

Tamiru *et al.* [21] reported that among all IBDV strains included in their study for phylogenetic comparison of VP2 nucleotide sequences, Ethiopian

strains form a cluster within the vvIBDV lineage. They have also shown that Ethiopian IBDV strains have mutations in the VP1 region.

**Pathogenesis of IBD:** To understand how the IBD virus adversely affects the chicken's immune system, relevant factors above early development immune system have to be understood. During embryonic development and through approximately 10 weeks of age, immune system cells (lymphocytes) travel to BF is be programmed to become antibody-producing cells [2]. If the IBD virus damages the BF in young chickens, the bursa of fabricus will not be capable of programming sufficient numbers of lymphocytes. Thus, the chickens will experience reduced immune system capabilities and immunosuppression [22].

The earlier the damage to the BF occurs; the lesser lymphocytes with antibody-producing capability will be programmed. Therefore, any IBD virus control program should attempt to protect the BF as early as possible.

In practical terms, if the BF can be protected against disease until at least 3 weeks of age with a chance for, an adequate number of lymphocytes to be programmed and the immune-suppressive effects of an IBD outbreak will be minimal [23].

**Incubation Period, Clinical Signs and Mortality:**

IBDV has a short incubation period of 2 to 3 days and the infection generally lasts 5 to 7 days. One of the earliest signs of IBDV infection is the tendency for birds to engage in vent picking. Clinical signs are described as acute onset of depression, trembling, white and watery diarrhoea, anorexia, prostration, ruffled feathers, vent feathers soiled with urates and hemorrhages in pectoral and thigh muscles. In severe cases, birds become dehydrated and in the terminal stages subnormal temperatures and death ensue. Naïve chickens between 3 and 6 weeks of age are most susceptible to the clinical form of IBD, which causes impaired growth, immune-suppression and mortality. Clinical signs are mainly characteristic of IBDV serotype I classic strains [24].

In fully susceptible flocks, mortality associated with infection due to classic strain may range from 1-60%, with high morbidity of up to 100%. A variant IBDV strains do not produce overt clinical signs, but cause immunosuppression and may cause mortality due to secondary opportunistic infections in immuno-compromised birds. In contrast, vvIBDV strains cause mortality of 50-60% in laying hens, 25-30% in broilers and 90-100% in susceptible leghorns. Susceptible chickens younger than three weeks of age may not exhibit clinical signs, but develop subclinical infections. This results in a decreased humoral antibody response due to B lymphocyte depletion in the cloacal bursa and a severe and prolonged immunosuppression. The most significant economic losses result from subclinical infections. This form of IBD infection greatly enhances the chicken's susceptibility to sequelae such as gangrenous dermatitis, chicken anemia virus, inclusion body hepatitis, respiratory diseases and bacterial infections [25].

**Subclinical and clinical IBD:** Infectious bursal disease follows one of two courses, depending on the age at which chickens are infected. The subclinical form of the disease occurs in chickens less than 3 weeks of age. Chickens present no clinical signs of disease, but experience permanent and severe immunosuppression. The reason young chickens exhibit no clinical signs of disease are not known. However, immunosuppression occurs due to damage to the bursa of Fabricius [26].

The majority of field infections are subclinical and this form is the more economically important form of the disease. Broiler integrations commonly have farms described as problem farms. Broilers grow on these farms typically have poor body weights and feed conversions, high mortality, excessive reactions to respiratory vaccines and high rates of condemnation at processing. In many cases, investigations have shown that these farms are heavily contaminated with the IBD virus. The poor performance of the broilers is due to factors relating to immune-suppression caused by subclinical IBD. The clinical form of IBD usually occurs in chickens from 3 to 6 weeks of age. The clinical disease has a sudden onset and the mortality rate in the flock increases rapidly. Clinical signs of disease include dehydration, trembling, ruffled feathers, vent pecking and depression. Affected chicken experiences a transient immune-suppression. On necropsy, the principle lesions are found in the BF [8].

**Pathology:**

**Gross Lesions:** Gross lesions observed in birds that are common to IBDV infection include dehydration, hemorrhage in breast and leg musculature, darkened discoloration of the pectoral muscles, occasional hemorrhages in the leg, thigh and pectoral muscles, increased mucus in the intestine and renal changes. The gross appearance of the kidneys may appear normal in birds that are necropsied during the course of infection. In birds that die or are in advanced stages of the disease, kidneys frequently show swelling and pallor with accumulation of urates in the tubules and ureters. The bursa of Fabricius is the predominant lymphoid organ affected by IBDV. Infections with classic strains of IBDV cause inflammation and hypertrophy of the bursa as early as day 3 post-infection. By day 4, the bursa is double its original size and weight due to edema and hyperemia. By day 5, the bursa returns to its normal weight, but continues to atrophy until reaching one-third or less of its original weight following day 8 post-infection. In contrast, variant strains of IBDV typically cause a rapid atrophy, mucosal edema and firmness of the bursa in the absence of inflammation. Only one variant isolate has been reported to cause bursal inflammation by day 2 or 3 post-infection, a gelatinous yellowish transudate covers the serosa surface of the bursa and longitudinal striations become visible. The bursa's normal white color shifts to cream and then, in some cases, gray during and following the period of atrophy. In addition, necrotic foci and petechial or ecchymotic hemorrhages on the mucosal surface may be observed in infected bursa [27].

Moderate to severe splenomegaly with small gray foci uniformly distributed on the surface has been reported. Occasionally, petechial hemorrhages have been in the mucosa at the junction of the proventriculus and gizzard. Compared to moderately pathogenic IBDV strains, vvIBDV strains induce similar bursal lesions, but cause more severe damages to the cecal tonsils, thymus, spleen and bone marrow [28].

**Microscopic Lesion:** IBDV infections produce microscopic lesions primarily in the lymphoid tissues i.e. cloacal bursa, spleen, thymus, cecal tonsils and Hardarian gland. Degeneration and necrosis of B lymphocytes in the medullary region of the bursal follicles is apparent within one day of exposure. Depleted lymphocytes are quickly replaced by heterophils, pyknotic debris and hyperplastic reticulo-endothelial (RE) cells. By 3 or 4 post-infection, IBDV-associated lesions are visible within all bursal follicles [29].

At this time, infections with classic IBDV strains have caused an inflammatory response marked by severe edema, heterophil infiltration and hyperemia in the bursa. Inflammation diminishes by day 4 post-infection (PI) and as necrotic debris is cleared by phagocytosis, cystic cavities develop in the medullary areas of the lymphoid follicles. Necrosis and infiltration of heterophils and plasma cells occur within the follicle, as well as, the inter follicular connective tissue. In addition, a fibroplasia the inter follicular connective tissue may appear and the surface epithelium of the bursa becomes involuted and abnormal [30].

Proliferation of the bursal epithelial layer generates a glandular structure of columnar epithelial cells that contains globules of mucin. During this stage of the infection, scattered foci of repopulating lymphocytes were observed; however, these did not develop into healthy follicles. Microscopic lesions caused by variant strains are characterized by extensive follicular lymphoid depletion and rapid atrophy of the cloacal bursa in the absence of an inflammatory response [31].

**Diagnosis of IBD:** Diagnosis of IBD involves consideration of the flock's history and of the clinical signs and post mortem lesions. Obviously, chickens less than 3 weeks of age present no clinical signs of disease, while chickens greater than 3 weeks of age present clinical signs as described. The severity of the clinical signs will depend upon the factors described. Confirmation of a diagnosis of clinical IBD can be made at necropsy by examining the bursa of fabricius during the early stages

of disease for characteristic gross lesions. During later stages of disease, it is difficult to confirm a diagnosis of IBD by examining only shrunken and atrophied bursa of fabricius [32]. In birds less than 3 weeks of age or in young chickens with maternal antibodies, IBD virus infections are usually subclinical. Thus, typical clinical signs are not present and diagnosis should be supported by histopathologic study of bursa of fabricius, serologic studies, or by virus isolation [31, 32].

**Identification of the Agent:** Clinical IBD has clearly characteristic signs and post-mortem lesions. A flock will show very high morbidity with severe depression in most cases lasting for 5-7 days. Mortality rises sharply for 2 days then declines rapidly over the next 2-3 days. Usually between 5% and 10% of birds die, but mortality can reach 30-40%. The main clinical signs are watery diarrhea, ruffled feathers, reluctance to move, anorexia, trembling and prostration. Post-mortem lesions include dehydration of the muscles with numerous ecchymotic haemorrhages, enlargement and discoloration of the kidneys, with urates in the tubules. The bursa of Fabricius shows the main diagnostic lesions [33].

In birds that die at the peak of the disease outbreak, the bursa is enlarged and turgid with a pale yellow discoloration. Intrafollicular haemorrhages may be present and, in some cases, the bursa may be completely hemorrhagic giving the appearance of a black cherry. Peri-bursal straw-colored edema will be present in many bursae. Confirmation of clinical disease or detection of subclinical disease is best done by using immunological methods as the IBDV is difficult to isolate [34].

For virus isolation, the methods described below should be followed. Differentiation between serotypes 1 and 2 or between serotype 1 subtypes or pathotypes should be undertaken by specialized laboratory serological tests. An AGID, VN or ELISA may be carried out on serum samples. The infection usually spreads rapidly within a flock of birds. Because of this, only a small percentage need to be tested in order to detect the presence of antibodies. If positive reactions are found in unvaccinated birds then it can be presented at the whole flocks are infected [10].

**Serological Tests:** Isolation of IBDV is not usually carried out as a routine diagnostic procedure. Specific antibody-negative chickens may be used for this purpose, as may cell cultures or embryonated eggs from specific antibody-negative sources [35]. However, some difficulty may be experienced in using the latter two systems as the virus does not readily adapt to them.

If successful, the identity of the virus can be confirmed by the virus neutralization (VN) test. The agar gel Immuno-diffusion (AGID) test can be used to detect viral antigen in the bursa of fabricius. A portion of the bursa is removed, homogenized and used as antigen in a test against known positive antiserum. This is particularly useful in the early stages of the infection, before the development of an antibody response. An immune-fluorescence test using IBDV-specific chicken antiserum can also be used to detect antigen in bursal tissue. Antigen-capture enzyme-linked immunosorbent assays (ELISAs) based on plates coated with IBDV-specific antibodies have also been described for the demonstration of viral RNA in the bursa of Fabricius [36].

**Differential Diagnosis of IBDV:** Acute coccidiosis (In coccidiosis there are sudden onset, ruffled feathers, bloody droppings, but no bursal lesion); nephrotic disease conditions (Caused by water deprivation and with nephrotoxic, but with no bursal lesion); hemorrhagic syndromes (Sulfa toxicity cause muscular and mucosal hemorrhages, but with no bursal lesion); mareks disease (Cause bursal atrophy but bursal lesions are very distinct) and adeno-virus infection (Atrophy of bursa, but intranuclear-inclusion bodies in hepatocytes and pancreases are characteristic in adenovirus infection) [37].

**Economic Significance of IBD:** It has been described throughout the world and its socioeconomic significance is recognized worldwide. The most economic significances of this disease are; higher mortality especially during initial outbreak, immune suppression, susceptibility and vaccination failure [38].

**Surveillance and Management:** Post outbreak hygiene measures may not be effective as the virus can survive for long periods in both housing and water. There are currently no formal surveillance programs for IBDV in wild birds in Australia. Poultry farmers, particularly backyard poultry farmers should be encouraged to protect their stock from exposure to wild birds to minimize risk of infection. An important Bio-security with adequate restriction to farm visits and distancing from other flocks. Pre-harvest food safety is important control point for the production of safe and healthy chicken food [39].

**Prevention and Control of IBD:** An effective IBD prevention and control program must involve an effective breeder vaccination program, an effective bio-security program and an effective broiler vaccination program. Immunization of breeders is an important part of the

IBD control program. Antibodies produced by the hen are passed through the egg to the broiler chick. These maternal antibodies, if present in adequate levels, protect the chicks against subclinical IBD. In following comprehensive breeder vaccination program is recommended: at 12 to 15 days of age - IBD live; at 85 days of age - IBD live or inactivated; and at 120 days of age -IBD inactivated. Revaccinate at 38 to 42 weeks of age with an inactivated IBD vaccine if titers are low [40].

Routinely monitor breeder IBD antibody titers to ensure vaccines are administered properly and that the chickens respond appropriately. Effective control of IBD in commercial broilers requires proper clean-up and disinfection between flocks and restricts traffic (People, equipment and vehicles) to the farm. The development and enforcement of a comprehensive bio-security program is the most in limiting losses due to IBD. Phenol and formaldehyde compounds have been shown to be effective for disinfection of contaminated premises [41].

Efforts at bio-security (Cleaning, disinfecting, traffic control) must be continually practiced. A third factor to consider in the IBD prevention and control program is vaccination of the broilers to prevent clinical IBD. Three categories of vaccines, based on their pathogen city, have been described: 1) mild, 2) intermediate and 3) virulent. The intermediate type IBD vaccines are most commonly used. These vaccines can stimulate earlier than the mild-type vaccines, without significant damage to the BF as may occur with the virulent type vaccines. The timing of broiler vaccination depends on the level of maternal antibody present in the chicks. High levels of maternal antibody at the time of vaccination will neutralize the vaccine virus. Thus, only a limited active immune response results and chickens will be susceptible to disease as maternal titers decrease [42].

If low levels of maternal IBD titers are present in the chicks, vaccination may not be effective on farms contaminated with virulent field virus. Approximately 10 to 12 days are required after vaccination for chickens to develop minimal protective titers. During this "lag time," chickens are susceptible to IBDV. In addition, virulent IBD virus's vaccine is able to break through higher maternal titers than milder vaccine viruses [43].

## CONCLUSIONS

Infectious bursal disease (IBD) is caused by a virus that is a member of the genus *AviBirnavirus* of the family *Birnaviridae*. Very virulent strains of classical serotype 1 are now common and are causing serious disease in many

countries. Mode of transmission is primarily through fecal oral route. Also Feed, water and poultry house litter become contaminated which cause IBD. Outbreak of suspected IBD case was reported to the National Veterinary Institute from a commercial poultry farm in Debre-Zeit town located 45 kms to the South of Addis Ababa and around Gondar. The infectious bursal disease (IBD) is recognized as an important disease of young chickens worldwide. It causes unthriftiness, anorexia, ruffled feathers, diarrhoea and mortality in affected flocks. An effective IBD prevention and control program must involve an effective breeder vaccination program, an effective bio-security program and an effective broiler vaccination program. Immunization of breeders is an important part of the IBD control program.

Based on the above conclusion, the following recommendations are suggested:

- Flock maternal antibody level of IBD should be checked in day old chicks (Sample test) to assess and determine vaccination schedule.
- Day old chicks should be purchased from replier hatcheries which routinely vaccinate breeder birds/parent stock.
- Very strict restriction should be imposed for entry of vehicles, equipments and visitors to this poultry farm. Effective bio-security should be in place.
- Following outbreak, this farm should be depopulated and completely cleaned and disinfected and cages, meshes should be flamed.
- Following disinfection using phenol and formalin, this house should be vacant for at least 6 months before stocking.

## REFERENCES

1. Muller, H., C. Scholtissek and H. Becht, 1979. The genome of infectious bursal disease consists of two segments of double stranded RNA. *Journal of Virology*, 31(1): 584-589.
2. Hitchner, S.B., 1970. Infectivity of infectious bursal disease virus. *Poultry Science*, 49: 511-516.
3. Kusters, J., H. Betch and R. Rudolph, 1972. Properties of infectious bursal agent of chicken (IBA). *Medical Microbiology and Immunology*, 157: 291-298.
4. Weisman, J. and S.B. Hitchner, 1978. Virus-neutralization versus agar-gel precipitin tests for detecting serological response to infectious bursal disease virus. *Avian Diseases*, 22: 598-603.
5. Cosgrove, A.S., 1962. An apparently new disease of chickens - avian nephrosis. *Avian Diseases*, 6: 385-389.
6. Ignjatovic, J.S. Sapats, R. Reece, A. Gould, G. Gould, P. Selleck, S. Lowther, D. Boyle, D.H. Jackwood and Y.M. Saif, 1987. Antigenic diversity of infectious bursal disease viruses. *Avian Diseases*, 31: 766-770.
7. Brown, M.D., P. Green and M.A. Skinner, 1994. Sequences of recent European "very virulent" isolates of infectious bursal virus are closely related to each other but are distinct from those of "classical" strains. *Journal of General Virology*, 75: 675-68.
8. Saif, M.Y., 2003. *Disease of poultry*. 11<sup>th</sup> ed. Iowa state press: Black well science, pp: 1070-1080.
9. Jackwood, D.J. and S.E. Sommer-Wagner, 2005. Molecular epidemiology of infectious bursal disease viruses: distribution and genetic analysis of newly emerging viruses in the United States. *Avian Diseases*, 49: 220-226.
10. Winterfield, R.W., A.M. Fadly and A. Bickford, 1972. Infectivity and distribution of infectious bursal disease virus in the chicken. Persistence of the virus and lesions. *Avian Diseases*, 16: 622-632.
11. Woldemariam, M. and A. Wossen, 2007. Infectious bursal disease: case report at region. *Ethiopian Veterinary Journal*, 11: 151.
12. Okoyo, J.O. and M. Uzoukwu, 2005. Infectious bursal disease among chickens. *Avian Dis.*, 25(4): 1034-103.
13. Sanchez, J.H. Stryhn, M. Flensburg, A.K. Ersbøll and I. Dohoo, 2005. Analysis of acute clinical infectious bursal disease in broiler flocks in Denmark. *Preventive Veterinary Medicine*, 71(3-4): 209-23.
14. Murphy, A.F., J.P.E.H. Gibbs, C.M. Orzine and J.M. Student, 1999. *Veterinary virology*. 3<sup>rd</sup> ed. London academic press, pp: 405-410.
15. Hailu, M., S.B. Tilahun and T. Negash, 2009. Incidence of infectious bursal disease in village chickens in two districts of Amhara Region, Northwest Ethiopia. *Livestock Research for Rural Development*. Volume 21, Article #214. Retrieved July 15, 2014, from <http://www.lrrd.org/lrrd21/12/maze21214.htm>



16. Jenberie, S., S.E. Lynch, F. Kebede, R.M. Christley, E. Gelaye, H. Negussie, K. Asmare and G. Ayelet, 2013. Genetic characterization of infectious bursal disease virus isolates in Ethiopia. *Acta Tropica*, Oct 18,130C: 39-43. doi: 10.1016/j.actatropica.2013.09.025.
17. Zeleke, A., E. Gelaye, T. Sorri, G. Ayelet, A. Sirak and B. Zekarias, 2005. Investigation on IBD disease outbreak in Debre-Zeit, Ethiopia. *International Journal of poultry science*, 7: 504-506.
18. Landgraf, H., E. Vielitz and R. Kirsch, 1967. Occurrence of an infectious disease affecting the bursa of Fabricius (Gumboro disease). *Dtsch Tierärztl Wochenschr*, 74: 6-10.
19. Saif, Y.M., 1998. Infectious Bursal Disease and Hemorrhagic Enteritis. *Poultry Science*, 77: 1186-1189.
20. Kassa, S.A. and W. Molla, 2012. Seroprevalence of infectious bursal disease in backyard chickens of North West Ethiopia. *Scientific Journal of Crop Sciences*, 1(1): 20-25.
21. Tamiru, N., G. Esayas, P. Henning, G. Beatrice and R. Silke, 2012. Molecular Evidence of Very Virulent Infectious Bursal Disease Viruses in Chickens in Ethiopia. *Avian Diseases Sep.*, 56(3): 605-610.
22. Pattison, M., D.J. Alexander and J.W. Harkness, 1975. Purification and preliminary characterization of a pathogenic strain of infectious bursal disease virus. *Avian Pathology*, 4: 175-187.
23. Müller, H. and N. Nitschke, 1987. The two segments of infectious bursal disease virus genome are circularized by a 90,000 Da protein. *Virology*, 159: 174-177.
24. Giambrone, J.J., D.L. Ewert and C.S. Eidson, 1977. Effect of infectious bursal disease virus on immunological responsiveness of chicken. *Poultry Science*, 56: 1591-1594.
25. Lukert, P.D. and S.B. Hitchner, 1984 Infectious bursal disease. M. S. Hofstad, H. J. Barnes, B.W. Calnek, W. M. Reid and H. W. Yoder, (Eds). *Diseases of Poultry*, 8th ed. Iowa State University Press, Ames, IA., pp: 566-576.
26. Jordan, P.M., D. Alexander and T. Farasher, 2002. *Poultry disease*. 5<sup>th</sup>.ed. London: W.B. Saunders, pp: 319-322.
27. Weiss, E. and I. Kaufer-Weiss, 1994. Pathology and pathogenesis of infectious bursal disease In: *Proc. International symposium on infectious bursal disease and chicken infectious anemia*, Rauischholzhausen, Germany, 21-24 June 1994.
28. Ashraf, S., G. Abdel-Alim and Y.M. Saif, 2006. Detection of antibodies against serotypes 1 and 2 infectious bursal disease virus by commercial ELISA kits. *Avian Diseases*, 50: 104-109.
29. Cheville, N.F., 1967. Studies of the pathogenesis of Gumboro Disease in bursa of Fabricius, spleen and thymus of the chicken. *American Journal of Pathology*, 51: 515-551.
30. Peters, G., 1967. Histology of Gumboro disease. *Berl Munch Tierarztl Wochenschr*, 80: 394-396.
31. Campbell, T.W. and E.H. Coles, 1986. Avian clinical pathology. In: Coles E.H. Ed., *Veterinary clinical pathology*. Philadelphia, PA, USA, WB Saunders, pp: 289-290.
32. Rajaonarison, J.J., S.M. Rakotonindrina, E.K. Rakotondramary and S. Razafimanjary, 2006. Gumboro Disease (Infectious bursitis) in Madagascar. *Revue d'élevage et de médecine vétérinaire des pays tropicaux*, 47(1): 15-17.
33. Khan, R.W., F.A. Khan, K. Farid, I. Khan and M.J. Tariq, 2009. Prevalence of Infectious Bursal Disease in broiler in district Peshawar *Journal of Agricultural and Biological Science*, 4(1): 1-5.
34. McNulty, M.S., G.M. Allan and J.B. McFerran, 1979. Isolation of infectious bursa disease virus from turkeys. *Avian Pathology*, 8: 205-212.
35. McFerran, J.B., M.S. McNulty, E.R. McKillop, R.M. Connemccracken, D.S. Collinn and G.M. Allan, 1980. Isolation and serological studies with infectious bursal disease viruses from fowl, turkey and duck: Demonstration of a second serotype. *Avian pathology*, 9: 395-04.
36. Marquardt, W.W., R.B. Johnson, W.F. Odenwald and B.A. Schlotthober, 1980. An indirect enzyme-linked immunosorbent assay (ELISA) for measuring antibodies in chickens infected with infectious bursal disease virus. *Avian Diseases*, 24: 375-385.
37. Kaufer, I. and E. Weiss, 1980. Significance of bursa of Fabricius as target organ in infectious bursal disease of chickens. *Infection and Immunity*, 27: 364-367.
38. Muller, M., R. Islam and R. Raue, 2003. Research on IBD the past, present and future veterinary micro-biology, 97: 153-165.
39. Westbury, H., 2004. Virus strains from a flock exhibiting unusually high mortality due to infectious bursal disease. *Australian Veterinary Journal*, 82(12): 1050-1060.

40. Sharma, J.M., I.J.M. Kim, S. Rautenschlein and H.Y. Yen, 2000. Infectious bursal disease bursal disease viruses: distribution of newly emerging viruses in the United States. *Avian Diseases*, 49: 220-226.
41. Wyeth, P. J. and N.J. Chettle, 1990. Use of infectious bursal disease vaccines in chicks with maternally derived antibodies. *The Veterinary Record*, 126: 577-578.
42. Box, P., 1989. High maternal antibodies help chickens beat virulent virus. *World's Poultry Science Journal*, 53: 17-19.
43. Godard, R.D., P.J. Wyeth and W.C. Varney, 1994. Vaccination of commercial layer chicks against infectious bursal disease with maternally derived antibodies. *The Veterinary Record*, 17: 237-274.